In the Specification:

Please replace the sequence listing (filed June 29, 2006) with the revised sequence listing filed herewith.

Please replace the paragraph at page 9, lines 5-8, with the following amended paragraph:

Figure 11 is a graph showing inhibition of IL-2 binding to its receptor, CD25, by supernatants of human monoclonal antibodies AB1, AB7, AB11, and AB12, compared to inhibition of IL-2 binding by Zenapax® antibody (daclizumab, recombinant humanized IgG1 anti-CD25 antibody, Roche).

Please replace the paragraph at page 9, lines 10-11, with the following amended paragraph:

Figure 12 is a graph showing inhibition of Zenapax® antibody binding to CD25 by human monoclonal antibodies AB1, AB7, AB11, and AB12.

Please replace the paragraph at page 9, lines 13-15, with the following amended paragraph:

Figure 13 is a graph showing inhibition of anti-CD3 antibody-induced T cell proliferation (using PBMCs) by human monoclonal antibodies AB1, AB7, AB12, compared to inhibition by a control antibody (hIgG1/κ) and Zenapax® antibody.

Please replace the paragraph at page 9, lines 17-19, with the following amended paragraph:

Figure 14 is a graph showing inhibition of MLR by human monoclonal antibodies AB1, AB7, AB12, compared to inhibition by a control antibody (hIgG1/κ) and Zenapax® antibody.

Please replace the paragraph at page 18, lines 6-17, with the following amended paragraph:

The percent identity between two nucleotide sequences can be determined using the GAP program in the GCG software package (available on the internet at the website at ~~http://www.~~gcg.com), using a NWSgapdna.CMP matrix and a gap weight of 40, 50, 60, 70, or

80 and a length weight of 1, 2, 3, 4, 5, or 6. The percent identity between two nucleotide or amino acid sequences can also be determined using the algorithm of E. Meyers and W. Miller (*Comput. Appl. Biosci.*, 4:11-17 (1988)) which has been incorporated into the ALIGN program (version 2.0), using a PAM120 weight residue table, a gap length penalty of 12 and a gap penalty of 4. In addition, the percent identity between two amino acid sequences can be determined using the Needleman and Wunsch (*J. Mol. Biol.* 48:444-453 (1970)) algorithm which has been incorporated into the GAP program in the GCG software package (available at http://www.gcg.com), using either a Blossum 62 matrix or a PAM250 matrix, and a gap weight of 16, 14, 12, 10, 8, 6, or 4 and a length weight of 1, 2, 3, 4, 5, or 6.

Please replace the paragraph at page 18, lines 18-29, with the following amended paragraph:

The nucleic acid and protein sequences of the present invention can further be used as a "query sequence" to perform a search against public databases to, for example, identify related sequences. Such searches can be performed using the NBLAST and XBLAST programs (version 2.0) of Altschul, *et al.* (1990) *J. Mol. Biol.* 215:403-10. BLAST nucleotide searches can be performed with the NBLAST program, score = 100, wordlength = 12 to obtain nucleotide sequences homologous to the nucleic acid molecules of the invention. BLAST protein searches can be performed with the XBLAST program, score = 50, wordlength = 3 to obtain amino acid sequences homologous to the protein molecules of the invention. To obtain gapped alignments for comparison purposes, Gapped BLAST can be utilized as described in Altschul *et al.*, (1997) *Nucleic Acids Res.* 25(17):3389-3402. When utilizing BLAST and Gapped BLAST programs, the default parameters of the respective programs (*e.g.*, XBLAST and NBLAST) can be used. See the internet at the website of ncbi.nlm.nih.gov ~~http://www.ncbi.nlm.nih.gov~~.

Please replace the paragraph at page 57, lines 10-13, with the following amended paragraph:

Sequencing: The VDJ-regions were sequenced after cloning in the pGEMT-Vector System II. Sequencing was performed at Baseclear (Leiden, Netherlands).
The sequences were aligned to germline V-gene sequences in Vbase available on the internet at the website mrc-cpe.cam.ac.uk ~~(www.mrc-cpe.cam.ac.uk/imt-doc/public/intro.htm)~~.

Please replace the paragraph at page 60, lines 24-38, with the following amended paragraph:

Inhibition of binding of biotinylated IL-2 to its receptor by supernatants of human CD25 monoclonal antibodies: In order to examine the extent of which human monoclonal antibodies block or inhibit IL-2 binding to CD25 96-well plates (Greiner) were coated overnight at RT with rhCD25 (100 ng/ml; R&D systems, MN, USA), whereupon non-specific binding was blocked by coating the plates with PBSTC for 1 hour at RT. After washing (3x) the plates with PBST, 100 µl of sample antibody (concentration range: 10, 33, and 100 ng/ml) was added. For comparison Zenapax® antibody was also added. After 10 minutes, rIL-2-biotin (50 ng/ml) was added (1.5 hours, RT). After washing the plates 3x (in PBST), plates were incubated with streptavidin-poly-HRP (dilute 1:10,000 from stock) in PBS, and 100 µl was added to each well (1 hour, RT). After washing the plates (3x in PBST), 10 mg ABTS (Roche) per 10 ml ABTS buffer (Roche) was made and 100 µl added to each well. After 20 minutes, absorption was read at 405 nm with an ELISA reader (EL 808, Bio-Tek Instruments).. Data show one out of two representative experiments. As shown in Figure 11, supernatants of human CD25 monoclonal antibodies AB1, AB7, AB11 and AB12 were able to inhibit binding of biotinylated IL-2 to CD25 more efficiently than Zenapax® antibody.

Please replace the paragraph at page 61, lines 1-14, with the following amended paragraph:

Inhibition of binding of Zenapax® antibody to CD25 by supernatants of human CD25 monoclonal antibodies: In order to examine the extent of which human monoclonal antibodies block or inhibit binding of Zenapax® antibody to CD25, 96-well plates (Greiner) were coated overnight at RT with rhCD25 (100 ng/ml; R&D systems, MN, USA), whereupon non-specific binding was blocked by coating the plates with PBSTC for 1 hour at RT. After washing (3x) the plates with PBST, 100 µl of sample (concentration range: 10, 33, and 100 ng/ml) was added. After 10 minutes, biotinylated Zenapax® antibody (5 ng/ml) was added (1.5 hours, RT). After washing the plates 3x (in PBST), plates were incubated with streptavidin-poly-HRP (dilute 1:10,000 from stock) in PBS, and 100 µl was added to each well (1 hour, RT). After washing the plates (3x in PBST), 10 mg ABTS (Roche) per 10 ml ABTS buffer (Roche) was made and 100 µl added to each well. After 20 minutes, absorption was read at 405 nm with an ELISA

reader (EL 808, Bio-Tek Instruments). Data show one out of two representative experiments. As shown in Figure 12, supernatants of human monoclonal antibodies AB1, AB7, AB11, and AB12 block Zenapax® antibody binding to CD25.

Please replace the paragraph at page 61, lines 18-20, with the following amended paragraph:

Human antibodies were tested for their ability to inhibit T cell proliferation using the T cell proliferation assay . For comparison Zenapax® antibody as well as an isotype control antibody (hIgG1/κ) were also tested.

Please replace the paragraph at page 62, lines 10-13, with the following amended paragraph:

As shown in Figure 13, human monoclonal antibodies AB1, AB7, and AB12 inhibited anti-CD3 antibody-induced T cell proliferation in a dose-dependent manner. The inhibition by the human antibodies was more efficient than by Zenapax® antibody. Data show one out of three representative experiments.

Please replace the paragraph at page 62, lines 16-27, with the following amended paragraph:

Human antibodies were tested for their ability to inhibit MLR using the MLR assay. For comparison Zenapax® antibody as well as an isotype control antibody (hIgG1/κ) were also tested. Human PBMCs (obtained in buffy coats from Dutch Red Cross Blood Bank, Utrecht, Netherlands) from two non-MHC-matching donors were diluted in RPMI 1640 (supplemented with 10% FCS (Wisent Multicell optimum C241), 2 mM L-glutamine, 50 IU/ml penicillin, 50 μg/ml streptomycin (all derived from Gibco BRL, Life Technologies, Paisley, Scotland)) to 2.0 x 10^6 cells/ml. PBMCs from the first donor were irradiated (2000 rads) and mixed (1.0 x 10^5 cells/well) with PBMCs from the second donor (1.0 x 10^5 cells/well) in 96-well flat bottom plates (Greiner) in triplet. Then, 50 μl of increasingly diluted experimental antibodies were added to the cells (ranging from 50 ng/ml to 0.8 ng/ml, in two-step dilutions). After six days of culture, (37 °C, 5% CO_2) proliferation was quantified by using BrdU (end concentration: 10 μM, Roche) according to the method described above.

Please replace the paragraph at page 62, lines 28-31, with the following amended paragraph:

As shown in Figure 14, human monoclonal antibodies AB1, AB7, and AB12 inhibited the MLR in a dose-dependent manner. Inhibition of MLR by AB1, AB7, and AB12 (at doses between about 1 and 3 ng/ml) was more efficient than inhibition by Zenapax® antibody. Data show one out of three representative experiments.

Please replace the paragraph at page 62, line 34 through page 63, line 8, with the following amended paragraph:

Affinity analyses were assessed by monitoring changes in surface plasmon resonance using a BIAcore 3000 instrument. A BIAcore 3000 and BIAcore 3000 software control (BIAcore, Uppsala, Sweden, lot#BR-1100-43) was used. Human CD25 (R&D Systems, lot#223-2A/CFO) was immobilized to a CM-5 sensor chip at low-density (BIAcore, lot#BR-1000-14) using amine-coupling chemistry according to the manufacturer's recommendations. After blocking the residual binding sites of the activated sensor chip using ethanol-amine-HCl, a kinetic analysis was performed at 25 °C (according to the manufacturer's recommendations) using human monoclonal antibody AB12 and for comparison Zenapax® antibody. Samples containing AB12 and Zenapax® antibody, respectively, were flowed over the surface of the coated sensor chip allowing AB12 and Zenapax® antibody to associate with rhCD25. The association and dissociation of AB12 and Zenapax® antibody, respectively, were monitored using surface plasmon resonance (SPR) on the sensor chip. The results were visualized using a BIAcore 3000 (Bio-tek Instruments) and analyzed using the BIAevaluation Software 3.1 (BIAcore, Uppsala, Sweden) and Languir binding 1:1 was used as pre-fixed model.

Please replace the paragraph at page 63, lines 11-12, with the following amended paragraph:

The K_D of Zenapax® antibody for the binding to rhCD25 determined by BIAcore analysis: $1.52 \times 10^{-10} \pm 0.27 \times 10^{-10}$.